

Femoral Anastomotic Aneurysms

LARRY H. HOLLIER, M.D., ROBERT C. BATSON, M.D., ISIDORE COHN JR., M.D.

AORTOFEMORAL GRAFTING has provided innumerable patients with relief from symptoms of arterial insufficiency and generally gives long lasting beneficial results. However, a small percentage of patients will have their successful results marred by the subsequent development of a femoral anastomotic aneurysm.^{2,5,11,28} Recent advances in peripheral vascular surgery have decreased the incidence of anastomotic pseudoaneurysms, but currently there is no way to eliminate this complication entirely.

Since 1974, the LSU Vascular Surgery Service has managed all vascular patients by one team of surgeons. A significant number of the patients referred to the service had pseudoaneurysms which developed in the femoral region after previous aortofemoral bypass. All of these early patients had their initial surgery at other hospitals or by other surgeons so the exact details of the original grafting procedures were unavailable. All pseudoaneurysms discovered were operated on and attempts were made to determine the cause and to prevent further similar problems. The findings and results of this study provide the basis of this report.

Clinical Material

From 1974 to the present, the LSU Vascular Surgery Service at Charity Hospital, Veterans Administration Hospital, and Hotel Dieu Hospital in New Orleans has managed 87 patients with femoral pseudoaneurysms. Six patients had traumatic femoral pseudoaneurysms, two occurring after gunshot wounds and four after angiographic procedures. Two patients had a femoral anastomotic aneurysm after a distal bypass and one patient had a femoral mycotic aneurysm. These nine patients are a separate problem and will not be discussed here.

Seventy-eight femoral anastomotic pseudoaneurysms occurred in 53 patients after aortofemoral bypass. Thirty-seven of these patients (69.8%) had a unilateral pseudoaneurysm while 16 patients (30.2%) had bilateral

From the Department of Surgery, Section of Vascular Surgery, Louisiana State University School of Medicine, New Orleans, Louisiana

occurrence. Five patients had nine recurrences of pseudoaneurysms after repair of their original pseudoaneurysms (Table 1).

The age ranged from 52 to 79 years. Thirty-nine patients, or 73.6%, had associated hypertension. Fifty-seven limbs (73.1%) had complete occlusion of the superficial femoral artery on the side of the femoral pseudoaneurysm.

Presentation

All patients had a mass palpable in the femoral area at the time of admission and in 65 patients this was the only presenting symptom. Two patients presented with a rapidly expanding mass in the groin and an obviously ruptured pseudoaneurysm. Eight patients presented with ischemic symptoms after sudden thrombosis of a pseudoaneurysm. Three patients were admitted for signs and symptoms of distal embolization from a femoral pseudoaneurysm with a patent superficial femoral artery. Most of the 53 patients underwent routine angiography to assess the status of the proximal inflow as well as distal runoff (Table 2).

Etiologic Factors

An attempt was made to evaluate the cause of the pseudoaneurysm in each case, though admittedly this essentially was a judgement made by the surgeon. Only one patient had fraying of the graft itself as the likely cause of the pseudoaneurysm. Two of the pseudoaneurysms had obvious infection as the cause; one of these was ultimately treated by obturator bypass, while the other had removal of the graft limb. Two patients had fractured polypropylene sutures as the apparent cause of the pseudoaneurysm: one had an intact Dacron® graft and a normal-appearing femoral artery with a dehiscence of the anastomosis and an evident broken polypropylene suture; another patient had an aorto-femoral graft on the right with a cross-femoral graft to the left leg, and a pseudoaneurysm at the Dacron-

Presented at the Annual Meeting of the Southern Surgical Association, December 3-5, 1979, Hot Springs, Virginia.

Reprint requests: Larry Hollier, M.D., Department of Surgery, Louisiana State University School of Medicine, New Orleans, Louisiana 70112.

Submitted for publication: December 7, 1979.

TABLE 1. Occurrence of Femoral Aneurysms in 53 Patients

Aneurysms	78
unilateral	37
bilateral	16
recurrent	9

to-Dacron anastomosis in the right groin, and again a fractured polypropylene suture was evident.

Five patients had a completely disrupted anastomosis with marked foreshortening of the limb of the Dacron graft. The graft appeared to have been "pulled" off the femoral artery which still appeared intact.

Fifty-seven pseudoaneurysms (73.1%) were thought to be due to host-vessel degeneration. Although some of those patients may have had hypertension, previous endarterectomy, or other associated factors, the specific finding was loss of integrity of the vessel wall. In some instances there was complete dissolution of the femoral artery in the area of the pseudoaneurysm.

In nine cases the records did not allow us to determine the cause of the pseudoaneurysm (Table 3).

Treatment

Early in this series surgical treatment consisted of simple resection of the pseudoaneurysm with reanastomosis if the graft could be pulled down into the groin without tension. Subsequently the policy was developed of routine interposition grafting with knitted Dacron and routine performance of an outflow procedure at the time of pseudoaneurysm repair. Twenty-three resections were done with direct reanastomosis while 55 had interposition grafts placed to avoid tension and provide for knitted Dacron anastomosis to the vessel. Thirty-three anastomoses were done without any specific outflow procedure. Forty-five anastomoses included outflow procedures, namely profundaplasty or superficial femoral artery angioplasty (32), and profunda angioplasty (9), or distal bypass (4) (Table 4).

Results

There were no deaths, no limb loss, and no early thrombosis resulting from the procedure. Except for the two cases of infected pseudoaneurysms, there were

no wound nor graft infections. Three patients, early in the series, who did not have angioplasty or other outflow procedure, have subsequently thrombosed that limb of the graft. Two were successfully treated by thrombectomy and profundaplasty, and one by thrombectomy, profundaplasty, and distal popliteal bypass.

Five patients developed recurrent pseudoaneurysms. Two patients had single recurrences and three had bilateral recurrences; one of the latter had an additional recurrence on one side. All but one of the secondary recurrences were treated by the addition of an interposition graft and an outflow procedure. The other patient had only an interposition graft placed after the first recurrence; she later had another recurrence and profundaplasty and distal bypass was performed at that time.

Discussion

Incidence and Cause

In 1965 Stoney, Albo and Wylie²⁴ reported 528 vascular reconstructive operations in which knitted Dacron grafts were used. Braided silk sutures were used for the anastomosis. Of 135 anastomoses to the common femoral or popliteal arteries, 32 aneurysms occurred, a per site incidence of 23.7%. In 1967 Sawyers, Jacobs and Sutton²⁰ reported a 10% incidence of false aneurysms developing in anastomoses to the common femoral or popliteal arteries. More recent reports by Szilagyi, Smith, Elliott, Hageman, and Dall' Olmo²⁸ in 1975 quoted a 3% per site incidence of pseudoaneurysm in the groin after aortofemoral grafting, and several other reports^{7,10,23} indicate an incidence of 2% or less at the present time. In our series, since the majority of our cases were referred from other institutions, we cannot provide an accurate incidence. However, based on our aortofemoral cases of the last five years, we estimate a 1% to 2% per site incidence at the present time.

In early cases braided silk sutures were used for most of the anastomoses.^{3,9,12,13,20,22,24-27} In 1940 Cutler and Dunphy⁸ demonstrated the degeneration of silk and its subsequent phagocytosis by monocytes, so this complication might well have been predicted. After reports^{2,3,7,9,13,20,24} of silk degeneration resulting in pseudoaneurysm formation became wide-spread, vascular surgeons switched to other suture material. It was soon noted that polyethylene sutures likewise resulted in fragmentation and a high incidence of pseudoaneurysms resulted.²³ Even polypropylene, as in two of the cases in our series, has shown evidence of fragmentation and breakage and is potentially the source of many aneurysms in the future. Of the sutures available today, braided Dacron is the suture

TABLE 2. Presenting Signs

	Number of Patients
Mass	65
Thrombosis	8
Embolization	3
Rupture	2

of choice for prevention of pseudoaneurysm formation.¹³ Moore and Hall¹³ stated, "The ultimate strength of a prosthetic vascular anastomosis is dependent on the suture material." This is based on the fact that there is no true healing between the prosthetic graft and the host vessel. More accurately, however, one must state that the ultimate strength of the anastomosis is the sum of all factors, namely, type of prosthesis, strength of the suture, host vessel integrity, mechanical factors, and rheologic factors. Although in the past pseudoaneurysm formation frequently resulted from failure of suture material, in modern vascular surgery one should now discount suture failure as anything but a minor etiologic factor in anastomotic aneurysms.

Numerous papers call attention to the rigidity of the Dacron prosthesis (especially the earlier types prior to 1960), the motion of the graft across the joint, and excessive traction on the anastomosis by a graft placed under tension as playing significant roles in the disruption of the femoral anastomosis.^{3,17,18,22} A rigid graft plus the movement of the graft across a joint would appear to produce shearing forces that cause late separation of the anastomosis.^{24,25} Even though diseased vessels are stiffer than normal arteries, they are still more compliant than the prosthetic grafts.^{17,21,26} The stress-strain characteristics in this situation therefore can place additional stress on the host vessel. Similarly, anastomosis with the graft under excessive tension would appear to place undue stress on both sutures and host vessel and could lead to anastomotic disruption. However, the modern, softer, knitted Dacron prostheses have much more flexibility and remain more pliable than the previous grafts, even across the hip joint.²⁷ Also, vascular surgeons have learned to leave enough length of the graft limbs to avoid excess tension. These, therefore, appear to be less frequent causes of anastomotic pseudoaneurysms today.

Szilagyi, Whitcomb, Schenker, and Waibel,²⁹ as well as others,^{11,14} have demonstrated the efficiency of end-to-end anastomoses, rather than end-to-side anastomoses, in providing the most hemodynamically suitable flow for the prevention of pseudoaneurysms. According to the laws of fluid flow relating to arterial grafting, the larger the angle between the graft and the recipient artery, the smaller the rate of flow through the anastomosis and the greater the turbulence and stress on the suture line.²⁹ These principles are well accepted today and most surgeons tend to make their femoral anastomoses with a small angle of incidence and a smooth, elliptical orifice. Although one does an end-to-side anastomosis for the routine aortofemoral bypass, an end-to-end anastomosis should be used for repair of pseudoaneurysms.

TABLE 3. Cause of Femoral Aneurysms

	Number of Patients
Graft failure	1
Infection	2
Suture line defect	2
Suture failure	2
Graft tension	5
Vessel degeneration	51
Undetermined	9

Wound complications, whether it be infection, seroma, or hematoma, all tend to increase drastically the incidence of anastomotic pseudoaneurysms.²⁸ Anti-coagulants should generally not be used postoperatively, since they tend to increase suture line leakage and result in hematoma and pseudoaneurysm formation.^{4,7,14,19} The use of preoperative antibiotics and intraoperative antibiotic irrigation can reduce wound and graft infection. Routine ligation of transected lymphatics in the femoral incision will prevent lymphocele and seroma formation. It would appear that careful attention to these details can, therefore, frequently eliminate these other causes of anastomotic pseudoaneurysms.

Despite the improvements in surgical technique, grafts, and suture materials, there still remains the spectre of unavoidable host vessel degeneration. Vessel degeneration has always played a primary role in anastomotic pseudoaneurysm formation, but is even more obvious now that other factors have decreased.^{2,4,6,15,22,31} Even early papers^{4,15,22} noted arterial wall deficiency as a leading cause of pseudoaneurysms, but tended to lessen its importance by citing hypertension, endarterectomy, and mechanical factors as etiologic entities. In actuality, most femoral anastomotic pseudoaneurysms are, in fact, due to host vessel degeneration.^{15,31} Endarterectomy can weaken the wall. Rigid grafts and various mechanical factors can add stress and likewise weaken the wall. Hypertension adds additional stress and can be an associated factor in vessel degeneration.³ They are all, however, merely associated or predisposing factors. In our

TABLE 4. Treatment

	Number of Patients
Repair without outflow procedure	33
Repair with outflow procedure	45
distal bypass	4
double angioplasty	9
single angioplasty	32
Interposition graft	55
No interposition graft	23
Neither graft nor angioplasty	17
Both graft and angioplasty	37

series, 57 of 78 pseudoaneurysms (73.1%) appeared directly attributable to vessel degeneration. We believe that the primary reason for host vessel degeneration is the increased "intra-anastomotic tension" that occurs from combinations of many factors, some of which have been mentioned. Even the sturdiest suture material will be ineffective if the tissue to which it is sewn is deficient. If we agree that the new knitted Dacron grafts do not fray as the older woven Teflon® or woven Dacron grafts did, and that the braided synthetic sutures have increased overall integrity, then it is obvious that the limiting factor becomes the host vessel itself if disruption is to occur. Infection can attack the anastomotic integrity, because the bacterial proteinase removes fiber from the anastomotic line and breaks down the seal with resulting hematoma formation and often expansion of this into a pseudoaneurysm.¹ Continued pressure can then cause progressive breakdown of the host vessel wall. An endarterectomized arterial wall consists of a very thinned-out layer of adventitia and this would, at least theoretically, suggest its ultimate deterioration with time and additional stress factors.

In reviewing the work of Szilagyi, Whitcomb, Schenker, and Waibel,²⁹ it is evident that the mechanical factor of turbulence can play a major role in increased intra-anastomotic tension. Although a properly constructed end-to-end anastomosis is virtually as efficient as an intact artery in transmitting blood, in the case of end-to-side anastomoses, the larger the angle between the graft and the recipient artery, the smaller is the rate of flow through the anastomosis. This "bend" in the conduit creates turbulence and loss of flow rate because of the disturbance in pressure head brought about by the abrupt change in the direction of the flow. In addition, a sharp-edged orifice, such as formed by an anastomosis through a simple slit in the arterial wall, produces a greater reduction in flow than when a bell-mouthed orifice is created.^{7,29} Christensen and Bernatz stated, "One can assume that the energy liberated during the sudden loss of flow rate, according to the above-named two factors, is in part distributed at the site of the anastomosis, thereby increasing the stress on the suture line."⁷ In similar fashion, occlusion of a superficial femoral artery with change in the angle of flow from a horizontal progression to a more acute angle would likewise cause increased anastomotic tension. When an elbow or bend occurs the differences in velocity and pressure head along the outer wall compared to the inner wall produce secondary currents, changing the rate of flow and the dissipation of energy to the anastomotic line. We find it interesting to note that 57 limbs had superficial femoral artery occlusion in our series. By reviewing

the above laws of fluid flow and arterial grafting, one should expect that the hemodynamic and pressure changes would result in increased tension at the level of anastomosis, since the angle of outflow is dramatically changed. In this context, we see that all of the many factors mentioned in previous articles can contribute to this increased intra-anastomotic tension and ultimate host vessel degeneration. Realistically, however, the end result is arterial degeneration and pseudoaneurysm formation.

Technique

Surgical intervention should be considered in all cases of anastomotic femoral pseudoaneurysm, regardless of size, since nearly 20% had some complication (hemorrhage, thrombosis, or embolization) related to the pseudoaneurysm.^{1,3,7,15,16,20,22,30} One should plan to resect the aneurysm and redo the entire anastomosis, rather than simply placing a few reinforcing sutures, even though there may be only partial dehiscence.^{1,22,24,30} Preoperative antibiotics and intraoperative antibiotic irrigation should be used to prevent infection. Meticulous attention to skin preparation and draping is mandatory. An incision is made directly over the pseudoaneurysm and exposure obtained at the inguinal ligament for proximal control of the Dacron graft. After exposing the anterior surface of the pseudoaneurysm, intravenous heparin is given and the mass is opened and the clot removed. Distal control is obtained by means of one or more balloon catheters. The profunda is not dissected until the pseudoaneurysm mass is decompressed so that one might improve visibility and lessen the risk of damage to the profunda and collateral vessels. Minimal dissection is done to minimize disruption of the supportive scar tissue in the area. The graft is then transected at the inguinal ligament and an end-to-end anastomosis is made between the graft and a new segment of knitted Dacron. The outflow vessel(s) is then opened longitudinally and the interposed Dacron segment is sutured in end-to-end fashion to the transected femoral artery with a "tongue" of the graft extended down the outflow vessel as a patch angioplasty. Braided synthetic sutures are obviously preferred on a theoretic basis. Heparin reversal is not used, but postoperative heparin is not given. The wound is closed in four layers with non-absorbable sutures and sterile dressings applied.

Occasionally, the angioplasty must be continued for an extended length down the profunda and the vessel may be rather attenuated. In that situation a vein patch is preferred and the graft is then sewn end-to-end to the patched vessel. If both the superficial femoral and profunda femoris arteries are open, a "double-tongued"

graft is fashioned and angioplasty performed on both vessels. If run-off at the femoral level is questionable, a distal bypass may be added to assure patency and help prevent recurrent pseudoaneurysms.

Summary

Both the literature and this experience support host vessel degeneration as the primary etiologic factor in femoral anastomotic aneurysms. Associated factors that produce increased "intra-anastomotic tension," such as hypertension, superficial femoral artery occlusion, and flow turbulence, appear to contribute to vessel deterioration. Other factors, much less prevalent in present-day vascular surgery, such as rigid grafts, deficient suture material, inappropriate angle of incidence, and excessive tension on the graft can contribute to anastomotic disruption.

Certain guidelines may be helpful in the management of femoral pseudoaneurysm. 1) Redo the entire anastomosis, rather than simply resuturing a disrupted edge. 2) Use minimal dissection to avoid injury to outflow vessels and to limit disruption of supportive tissue. 3) Use braided synthetic suture material. 4) Avoid tension by interposing a segment of graft between the proximal graft limb and the host vessel. 5) Use knitted Dacron for the interposed segment so the new anastomosis to the host vessel will be with softer, more pliable fabric. 6) Assure smooth adequate outflow by end-to-end anastomosis with a patch angioplasty or distal bypass.

These guidelines should lead to a safe, reliable solution to one of the vexing complications of aortofemoral bypass procedure.

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DISCUSSION

DR. JAMES D. HARDY (Jackson, Mississippi): Turning to these femoral anastomotic false aneurysms, which we all have, I think I would agree that degeneration of the artery itself is the most common cause of the development of these lesions. Incidentally, my colleague, Dr. J. W. Williamson, looked up our numbers, and they are in the sixties.

It should be noted—I don't know that it was emphasized—that in 30% of Dr. Cohn's patients the lesion was bilateral, and this is perhaps additional evidence that the cause is probably not a technical matter, but is in fact degeneration of the arterial wall.

Without repeating many of the excellent points made by Dr. Cohn a minute ago, I would say that two things which are especially